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HEALTH BILLS PENDING IN CONGRESS*

Part Four

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JULIAN HUXLEY once wrote, "To speculate without facts is to attempt to enter a house of which one has not the key." The desire to avoid speculation and obtain factual information on pending health legislation seems to be strong, judging from the fact that some of the bills are on the best-seller list of the Superintendent of Documents in Washington.

Important organizations, representing large groups of the population, have officially endorsed the major legislative proposals now in Congress. Many people from all walks of life favor government action in the whole field of health service, on the ground that protection of the right to health care is a fundamental function of any society aspiring to a genuine and rational economy of human resources and values.

ATTITUDE OF THE MEDICAL PROFESSION

In general, the medical profession supports the hospital survey and construction program embodied in the Hill–Burton Bill; the expansion and improvement of general public-health, maternal and child health services proposed in Title I of the National Health Bill; disability insurance as recommended in the Wagner–Murray–Dingell Bill of May, 1945; and federal aid for medical research and education as proposed in the National Science Foundation Bill, the Mental Health Bill, the National Health Bill and other pertinent measures.

But the physicians are not unanimous in their opinions on pending health legislation. A few insist that they are against any plan, and resent any encroachment on what they believe to be their own territory. Others prefer to pass responsibility to the economists by maintaining that if the standards of economic independence of the poor and the indigent are raised, the problem of medicine will take care of itself. Still others doubt that any plan can be a success so long as certain evils are permitted to persist. A physician recently wrote to Senator Pepper, "As long as the United States is a place of easy divorce, quick-lunch counters and horn-tooting automobile drivers, and as long as the Smiths try to keep up with the Joneses, this will be a land of constipation, stomach ulcers, coronary thrombosis and emotional instability, and I don't think any 'plan' is going to help a great deal."[1]

Many are willing to accept government participation in the organization of medical care, provided that it is confined to service for the control of communicable diseases, care of the mentally sick and defective and programs for the needy and special groups, such as the veterans. Here and there voices of doubt can be heard questioning the wisdom of supporting the legislative proposals officially endorsed by organized medicine. Some writers point out that the Hill–Burton Bill leaves the door ajar for serious federal interference in local affairs. Some physicians fear that the Kilgore Bill would restrict the traditional freedom of scientific research by bringing politics into science.

What is of paramount significance is the unqualified opposition of the American Medical Association to compulsory health insurance, as proposed in the National Health Bill, and to a system of tax-supported medical care, as recommended in the Maternal and Child Welfare Bill. This attitude on the part of physicians is not universal. Two organizations of physicians, composed of members of the American Medical Association, have repeatedly gone on record in favor of compulsory health insurance. Early in 1946, the Committee on Medicine and the Changing Order of the New York Academy of Medicine stated its belief thus: "It may be desirable to conduct careful experiments at state and local levels with compulsory government insurance, so that we may have in the near future comparative experience with the relative values of voluntary and compulsory procedure."[2]

*This is the last of a series of four lectures on medical sociology given at Harvard Medical School during February and March, 1946. They were sponsored by the Department of Preventive Medicine and were primarily intended for third-year students.

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Much of the opposition to legislative proposals dealing with medical care is due to misinformation concerning the contents of the bills, unfavorable personal experiences with some program, fear of a revolutionary change in the traditional pattern of practicing medicine and unfamiliarity of physicians with the history of organized care of the sick. As John Locke once wrote, "New opinions are always suspected and usually opposed, without any other reason but because they are not already common."

In the center of the debate at present is the method of compulsory health insurance. At the risk of laboring the obvious, it must be stated that health insurance by law is organized self-help financed by money that the self-supporting people are asked to save for the purpose of paying for their own care. What is changed by such legislation is the method of raising funds for the support of professional and institutional services. Instead of paying if and when service is received and in proportion to the amount of service rendered, people are required to pay in advance small average amounts, in return for which they are entitled to any or all of the services available under the program. What is not changed by such legislation is the institution of private practice of medicine, dentistry and related professions, the institution of voluntary hospitals and the institution of voluntary health agencies. Being, as it is, a method of organizing payment for medical care, compulsory health insurance is neither a cure-all for social evils nor the gateway to the millennium.

There is nothing new about compulsory health insurance. It was adopted in this country as early as 1798, when the Act for the Relief of Sick and Disabled Seamen was passed. At that time this method of raising money was considered the most equitable one, as Mr. Williamson stated in the House of Representatives. At present compulsory health-insurance programs are in operation in some thirty countries in Europe, Asia, South America and Australia, and in Mexico. The potentialities and limitations of this method are known to every student of the subject, and experience is more than ample. If Congress should pass the pertinent measure, it would be possible to avoid, rather than to repeat, the many mistakes made in sixty years of trial and error. With the knowledge now available a plan can be designed that provides not only for the organization of payment but also for the establishment and maintenance of high standards of service throughout the country.

The arguments advanced against the compulsory-health-insurance title of the Wagner–Murray–Dingell Bill and against the Maternal and Child Welfare Bill are many and varied. Those based on misinformation need no discussion here. Of the others, two deserve special consideration — the assertion that socialized medicine is just around the corner, and the assumption that so-called "political medicine" is about to be imposed on the American people.

Socialized Medicine

The term "socialization," and with it the term "socialized medicine," have been used in a shifting sense. The latter term actually has two quite dissimilar meanings.[2] It may denote a system of medical care organized on the basis of the political and economic theory of socialism. Such a program calls for public ownership of all facilities for medical care, with the abolition of nongovernmental ownership of institutions; for the employment of all professional personnel by public agencies, with the discarding of private practice; and for public administration of all health services, with elimination of nongovernment agencies. None of the bills under consideration propose the adoption of such a program.

The term "socialized medicine" may also mean a program of medical care organized for the purpose of adapting medicine to social needs and uses, with no regard to socialist doctrines and methods. In this sense, American medicine has been socialized for some time and to a considerable extent. At present, about three fourths of all beds in registered hospitals and probably about half of all clinics are in institutions built from tax funds and operated by local, state and federal agencies. Many and diverse socioeconomic groups are entitled to medical care at the taxpayers' expense. They include several million recipients of public assistance and many millions of self-supporting people, such as the veterans, merchant seamen, Indians and Eskimos, wives and infants of enlisted men, the medically needy and certain employees of the federal government. In the fiscal year 1943–1944, the total expenditures of tax funds for medical care of civilians amounted to at least $900,000,000. The workmen's compensation acts, which are in force in all the states but one, require medical care to be furnished to those covered, and in 1943–1944 $140,000,000 was spent for this purpose.

Taxation and insurance by law have been the two methods employed to finance medical-care programs in the past, and these are the very methods proposed for the organization of payment for medical care in the future.

The bugaboo of socialized medicine can frighten only those who mistake "social" for "socialistic." It is about time to stop fighting over terminology and to concentrate time and effort on the intricate task of working out the method of action that best promises to achieve the ends desired by all.

Political Medicine

The second argument, which expresses a fear of political interference, if not political control, derives from the fact that in the past the selection of patients, the appointment of personnel and the

methods of institutional administration were in some instances influenced by the desire to make friends, gain votes and reward favorite sons (and daughters) for faithful political activities. But the picture is not so black as some persons would have one believe. There are many examples of good government in the field of health administration. What is more significant, a growing number of agencies and communities have been making constant and determined efforts to clean their own houses and to eliminate the taint of political medicine.

The danger of political manipulation of a nation-wide system of medical care cannot be lightly dismissed, but it can be averted. Let us now resolve to establish the tradition of good government in every community. If we fail in this task, we shall be admitting our unwillingness to exercise one of our precious constitutional rights and duties.

The major bills now under consideration have one element in common: they require all appointments to administrative positions to be made strictly on the basis of merit. Enactment of these measures will go a long way toward eliminating political interference in the administration of health services. It will afford a unique opportunity not only to deprive the last politicians of a happy hunting ground but also to generalize professional direction and supervision of professional matters.

Voluntary Health Insurance

Voluntary health insurance as an alternative to a compulsory program has been repeatedly recommended by the American Medical Association. In 1938, this organization announced its support of voluntary cash-indemnity insurance plans to cover, in whole or in part, the costs of emergency or prolonged illness. At the same time the Association recommended the expansion of workmen's compensation laws to provide for meeting the costs of illness sustained as a result of employment in industry, and thus endorsed the principle of required insurance for occupational diseases. In June, 1945, a so-called "Constructive Program for Medical Care" was adopted. One of its fourteen recommendations called for the development in or extension to all localities of voluntary sickness-insurance plans and provision for the extension of these plans to the needy under the principles already established by the American Medical Association. In December, 1945, the House of Delegates adopted a resolution calling for the nation-wide organization of locally administered prepayment medical plans sponsored by medical societies, which would, it was held, achieve all the objectives of the Wagner–Murray–Dingell Bill with far less expense to the people and would provide the highest type of medical care without regimentation. In February, 1946, the Board of Trustees issued a ten-point restatement of the Association's previously announced fourteen-point program.[4] Standards for the acceptance of prepayment plans were established. A detailed plan for the development of the voluntary health-insurance program was announced to be in process of being worked out by a subsidiary organization known as Associated Medical Care Plans, and a new unit, the Division of Prepayment Medical Care Plans, was created to administer the voluntary plans under American Medical Association auspices.

The question is justified whether past experience warrants the belief that the American Medical Association can accomplish its objectives.

In 1945, about 2,600,000 persons were enrolled in so-called "medical-society plans." They accounted for approximately half the total membership of voluntary prepayment plans covering physicians' services. At a time when purchasing power was at an all-time high point, only an insignificant fraction of the population had decided to take advantage of the services offered by medical societies. Although the plans had been in operation for several years, some of them had failed to attract substantial numbers of subscribers. A case in point is the Western New York Medical Plan in Buffalo. This plan, established in 1940 and serving six counties with large populations, reported 40,000 subscribers as of the middle of 1945. With the exception of the programs in Oregon and Washington, the great majority of the medical-society plans offer extremely limited service. In 1945, two thirds of the persons enrolled were eligible only for surgical service in hospitals, and in many instances this service was restricted by ceilings on eligibility, the period of care or the total amount of cost. Only one person in seventeen was eligible for physicians' care at the home, office and hospital. In short, the majority of the medical-society plans are distinguished by the fact that exclusions exceed inclusions.

The eligibility requirements often contain a clause barring from the prepaid service persons and families with incomes above the comfort level. In some instances this level is set as low as $1500 for a single person and $2500 for a family. By establishing income limits, the majority of the medical-society plans have insured the poorer risks and disregarded the more favorable ones, thus acting contrary to the basic principle of insurance, have made the proposition unattractive to large families, management and labor and have placed the participating physicians in the unenviable position of investigators who have to check the family income to detect the patients who earn more than the limit.

The total cost of medical-society plans varies too widely to allow generalizations. The most successful plan, the Michigan Medical Service, in the middle of 1945 charged monthly rates of $0.70, $1.60 and $2.25 for single subscribers, a family of two and families including all unmarried children up to nineteen years of age, respectively. These prepayments covered surgical service in the hospital and some

ancillary services. The physicians were free to make additional charges to those whose average annual income exceeded $2000 in the case of single persons and $2500 in the case of families. The Michigan Hospital Service, an outstanding Blue Cross Plan, charged monthly rates of $0.80, $1.80 and $2.00, respectively, for ward care and $1.00, $2.20 and $2.40 for semi-private care. To qualify for fairly complete hospital care and surgical service in the hospital, a single person had to make monthly prepayments ranging from $1.50 to $1.70, and a family of three or more had to pay in advance from $4.25 to $4.65 a month. These amounts equaled 1.2 and 1.4 per cent, respectively, of the income of single persons earning $1500 a year, and 2.5 per cent and 2.8 per cent, respectively, of the annual income of families earning $2000. It must be borne in mind that in 1945 an annual income of $2000 was barely sufficient to cover the ordinary living expenses of the average city family with three members. Substantially higher rates and larger proportions of the income would be needed to extend the scope of the services. High prepayment rates, however, defeat their own purpose, since only a small fraction of the population can afford to pay them.

Most of the medical-society plans in actual operation discourage preventive medicine by excluding health examinations, by using deterrents, such as the deductible clause, and by confining medical care to catastrophic illness instead of trying to reduce, if not prevent, the occurrence of serious conditions by providing for early diagnosis and early treatment of disease. Limited in scope as those plans are, they cannot foster psychosomatic medicine. In many cases a premium is placed on surgery, little effort being made to establish and enforce standards covering the competence of those rendering the service for which payment is claimed.

In summary, many medical-society plans are organized merely for the purpose of making financial arrangements for the payment of doctors' bills. Little attention can be paid to the improvement of the quality of service. Judging from the numerous articles appearing in medical journals, many of the physicians participating in these plans are dissatisfied and are looking for "something else." Not a few show keen interest in cash-indemnity plans, although the shortcomings of this method have been proved time and again. In California, the Alameda County Medical Society, in opposition to the state medical society, recommends such a plan, and other county societies are said to have voiced their approval. In Wisconsin, the state medical society has just made an agreement with commercial insurance companies, which are to underwrite a limited indemnity policy, and many physicians are beginning to wonder why the medical society should "sell out" to companies organized for profit.

The policies adopted by medical societies in the past were dictated by necessity. The physicians drawing up the prepayment programs entertained no illusions about the impossibility of establishing at reasonable cost a voluntary health-insurance program providing for complete service, offering unlimited free choice of physicians and paying the participating physicians on a fee-for-service basis. Societies that tried it failed. In California and Michigan, the medical societies had to drop their original full coverage contracts because of heavy financial losses. Between the Scylla of excessively high prepayment rates covering complete service and full payment for professional services and the Charybdis of moderate prepayment rates for complete service with extremely low fees for the physicians, the medical societies chose the only safe way — the offer of inexpensive and limited service to the subscribers and the promise of full payment to the physicians.

These facts, stated and commented on in countless publications in medical journals, indicate that the American Medical Association has taken on a Sisyphean task. It seeks to develop a satisfactory national health-insurance program by voluntary action, although the natural limitations of the voluntary method are well known. It desires to achieve the goal of country-wide organization, although the rank and file of the physicians are divided concerning the basic principles to be applied. Stormy debates are going on over the advantages and disadvantages of cash-indemnity plans and service plans, of combination and separation of professional services and hospitalization, of complete coverage and partial coverage of service, of rigid and liberal eligibility requirements and of individual practice and group practice. The American Medical Association believes that it can make the enactment of a health-insurance law superfluous by promising something better in the future, although a broad and balanced health program is needed now.

* * *

Whatever the fate of the various health bills may be, the wide discussion of the proposals will set in motion many changes in community planning for health services in general and for more effective organization of hospital and professional services in particular.

The problems we are now facing are highly complicated. Their solution requires accurate analysis of facts, correct appraisal of existing medical-care programs, fearless use of reason, constructive criticism of any proposal for the improvement of health services and single-mindedness in carrying out what appears to be a sound policy and one that is in accord with democratic ideals.

As Franklin Delano Roosevelt said in his Jefferson Day Address, written shortly before his death: "The only limit to our realization of tomorrow will be our doubts of today. Let us move forward with strong and active faith."

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INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM IN INFECTIOUS MONONUCLEOSIS*

A Report of Two Cases

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INFECTIOUS mononucleosis is generally regarded as a benign disease having a uniformly good prognosis. In the majority of cases this belief is substantiated, but in occasional cases this disease causes permanent damage and sometimes seriously threatens life.

The clinical features of infectious mononucleosis have been described in detail[1–6] and there are numerous articles on the hematological findings.[1, 2, 5, 7–10] There have, however, been relatively few reports dealing with the abnormalities of the central nervous system sometimes encountered.

The first case of involvement of the central nervous system in infectious mononucleosis was reported by Johansen[11] in 1931. His patient presented meningismus, drowsiness, impaired memory, dysarthria and weakness, in addition to the usual anginous type of infectious mononucleosis. In the same year Epstein and Dameshek[12] reported a comparable case in which there was ptosis of the right eyelid, a positive Kernig sign, a transiently positive Babinski sign, blurred vision and delirium. Both these patients made complete recoveries within two months.

In 1935, Fledelius[13] reported a case of infectious mononucleosis complicated by diplopia due to paresis of the left inferior rectus muscle, associated with stiff neck, headache, exaggerated knee jerks and questionable plantar reflexes. Improvement was gradual, with complete recovery in eighty days. In the following year, Sucher and Schwarz[14] described 2 severe cases in which death seemed imminent. The first patient presented drowsiness, horizontal nystagmus, right oculomotor paralysis, ataxia, adiadokokinesis and positive Babinski, Oppenheim and Kernig signs. Improvement in the third week was followed by relapse in the fourth week, with convulsions, left hemiplegia, cyanosis and pulmonary edema. Recovery was effected after seventy-five days. In the second case there was coma, opisthotonus, severe convulsions, left hemiplegia and pulmonary edema, but recovery was rapid, being complete in three weeks.

The second American case was reported in 1937 by Davidsohn.[15] This patient had coma and convulsions. It is noteworthy that this was the first case of encephalitis due to infectious mononucleosis to be proved by the heterophil agglutination reaction, the diagnosis in the previous cases having been based on the combination of lymphadenopathy, lymphocytosis and the finding of the typical cells in blood smears.

In 1937, Gsell[16] reported 5 cases showing involvement of the central nervous system. One patient developed an optic neuritis, the remnants of which were visible a month later. Transient facial paralysis was present in 2 cases. In one of these, a right facial paralysis was followed a week later by involvement of the left facial nerve. In the other case, a right facial palsy lasting a week occurred a month after apparent recovery from an otherwise typical infectious mononucleosis.

Cases of infectious mononucleosis with involvement of the central nervous system have been reported more frequently in recent years. Various parts of the nervous system are primarily involved in different patients, so that the clinical picture may be that of meningitis,[17–19] encephalitis,[20–22] meningoencephalitis,[23] encephaloneuronitis,[24] encephalomyelitis[25] or neuronitis.[26, 27]

The time interval between the onset of infectious mononucleosis and the development of neurologic abnormality is quite variable. If one excludes headache† as a significant symptom, it is seen that involvement of the central nervous system may be apparent as early as the first day of illness[12] or be delayed until the thirty-first day,[16] but it usually occurs in the second week of illness. The signs and symptoms depend on what portion of the nervous system is attacked, and there therefore is considerable variation from one case to another. Table 1 lists the signs and symptoms recorded in the cases reviewed and gives the number of cases in which each finding occurred.

The cerebrospinal-fluid findings parallel the neurologic process; a pleocytosis accompanying meningitic signs and a normal or slight increase in cell count is found in neuronitis and in purely encephalitic cases. The cells are predominantly

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†Headache was present in the majority of cases, but is so frequent in apparently uncomplicated cases[5,28] that it is of little diagnostic value unless it is unusually severe.

lymphocytes. The protein content is usually increased, and the Pandy test may be strongly positive,[24] although it is sometimes negative.[12] The mastic curve is variable.[16, 24]

Little is known of the actual lesions in these cases, because there has been such a paucity of material for histologic study. Ziegler[29] has recently reported a case of infectious mononucleosis in which death resulted from rupture of the spleen. At autopsy, focal lesions were found in the liver and kidneys, with mononuclear infiltration, proliferation of reticulocytes and necrosis suggesting an acute infectious granulomatous process. Ziegler states that the disease appears to be a generalized infection, with specific localization in one or more of the tissues or organs of the body.

TABLE 1. *Signs and Symptoms of Involvement of the Central Nervous System in Infectious Mononucleosis.*

Sign or Symptom	No. of Cases	Sign or Symptom	No. of Cases
Headache	30	Absent abdominal reflexes	3
Stiffness of neck	17	Nystagmus	3
Positive Kernig sign	8	Opisthotonus	3
Lethargy	7	Paralysis of serratus anterior	2
Muscle twitching	6	Diplopia	2
Dizziness	6	Positive Oppenheim sign	2
Dysarthria	6	Oculomotor paralysis	2
Positive Babinski sign	5	Hemiplegia	2
Facial paralysis	5	Stupor	2
Nausea	5	Coma	2
Photophobia	4	Delirium	2
Convulsions	3	Optic neuritis	2
Ptosis	3	Absent cremasteric reflexes	2
Ataxia	3	Anosmia	1
Dysphagia	3	Blurred vision	1
Vomiting	3	Impaired memory	1
Adiadokokinesis	3	Paraplegia	1
Hyperesthesia	3	Quadriplegia	1
		Paralysis on upward gaze	1

*Prepared from the available data of 32 cases reviewed (including the author's cases).

In 1939, Thomsen and Vimtrup[30] reviewed 500 cases of infectious mononucleosis seen in Copenhagen and described 6 fatal cases. In 4 of these there was no associated disease, death being ascribed to infectious mononucleosis; in all 4 cases death was attributed to respiratory paralysis. In 1 case an autopsy was performed and the histologic study showed severe degeneration of the cells in the region of the respiratory center. With the notable exception of these cases, there have been no deaths from damage to the central nervous system by infectious mononucleosis.

Permanent residuums due to infectious mononucleosis are unusual, but they have been reported by Marchal[31] (anosmia) and Saksena[27] (paralysis of serratus anterior).

The following case reports illustrate the encephaloneuronitic and encephalomyelitic types of infectious mononucleosis. In both these patients there was evidence of permanent damage to the nerve tissue. The second case is also remarkable in that it represents one of the few reported cases of infectious mononucleosis in a Negro.[32, 33]

CASE 1. A 31-year-old man was admitted to the hospital on August 25, 1944, complaining of headache, chills and fever. He had previously been well in all respects until 3 days prior to admission, when he developed swelling below the left ear, malaise and a fever while on a railroad journey. During the next 2 days these symptoms increased in severity, headache occurred, and the patient was seen by a medical officer, who prescribed sulfonamide tablets. The patient took a total of 4 tablets within a period of 12 hours, but the fever, headache and malaise increased and were accompanied by chills, so that as soon as the train reached its destination he was admitted to this hospital. The past history and family history were noncontributory.

Physical examination on admission showed an acutely ill, flushed patient perspiring freely. The temperature was 103°F., the pulse 100, and the respirations 35. The significant findings were diffuse injection of the conjunctivas, slight reddening of the throat, and moderate enlargement of the left postmandibular lymph node, which was nontender. The blood pressure was 105/80. The heart and lungs were normal. The neurologic findings were normal.

The laboratory data are given in Table 2. Blood Kahn reactions were repeatedly negative, as were x-ray studies of the chest and skull. Serum from blood samples taken on September 11 and October 4 were sent to the Army Medical School, where they were found to be negative for the viruses of Western equine encephalitis, St. Louis encephalitis and lymphocytic choriomeningitis.

The temperature spiked to 103°F. on the 2nd and 3rd days and then dropped to normal, where it remained on September 1 and 2. It rose to 102°F. on September 3, and there was a low-grade fever until September 8, when the temperature became normal and remained so. The respiratory rate was normal after the 1st day, and the pulse was proportionate to the temperature.

On symptomatic treatment, the patient seemed to improve, but on August 30 considerable increase in the lymphadenopathy was noted, the cervical, supraclavicular, epitrochlear and popliteal nodes being palpable. On the night of September 2, the 11th day of the illness, the patient developed some difficulty in speech and chewing and stated that his right cheek felt as though it were asleep. These symptoms progressed and extended, coincident with the rise in temperature, and on September 4 examination showed slurred speech, inability to chew, complete bilateral facial paralysis and loss of the gag reflex. The eyes turned upward and slightly to the left when the patient attempted to close them, and there was paralysis of upward gaze without loss of convergence. The right abdominal reflex was sluggish, and the right cremasteric reflex was absent. The sensorium, deep reflexes and cerebellar functions were normal.

The chief complaint at that time was an overwhelming feeling of tiredness. The patient's condition was considered quite serious. Suction apparatus was required to keep the throat and air passages clear, and special nurses were assigned. An additional finding, the true significance of which was in question because of its close similarity to a hysterical tic, was the apparently involuntary sucking, smacking and mumping noises that the patient made with his mouth every few seconds. This phenomenon persisted for 1 week and then gradually disappeared. The gag reflex returned on September 6, and by the next day the lymphadenopathy was generally diminished and the marked fatigue had subsided.

During the next 3 days the patient complained of sharp pains at odd intervals in the posterior cervical and sternocleidomastoid muscles, weakness of which was demonstrated for 2 months. Between September 8 and 11, a small patch of diminished sensitivity to pain developed on the neck just below the ear, first on the right and later on the left. This persisted until September 21. The chewing and swallowing gradually improved, so that by September 15 the patient was eating soft foods without assistance. It was not until September 29 that he was able to eat toast, since his tongue, lips and masseters were so poorly co-ordinated that he frequently bit his lip, tongue or cheek. His speech improved considerably, although it remained explosive at times and vocalization was preceded by inspiratory stridor.

On October 19, the patient was transferred to a general hospital. The significant findings on October 21 were as follows: complete facial paralysis; loss of taste over the anterior two thirds of the tongue; hippus of both pupils; loss of pain and temperature sense over the distribution of the trigeminal nerves; generalized muscle weakness; cogwheel rigidity of the arms; marked weakness of trapezius and sternocleidomastoid

muscles; loss of the left superficial abdominal and cremasteric reflexes, with diminution on the right; and a hopping gait.

In November, the patient was transferred to another general hospital. Examination on November 12, the 82nd day of the illness, showed numerous abnormalities. The left leg dragged on walking, and the patient tended to fall toward the left with his eyes open or closed. There was adiadokokinesis, paralysis of upward gaze; loss of pain, vibratory sense, temperature perception and light touch throughout

venereal disease. The only previous hospitalization was in September, 1944, and was due to scabies and pityriasis rosea. At that time peripheral arteriosclerosis was also diagnosed, being manifested by intermittent claudication and x-ray evidence of calcification in the femoral and popliteal arteries. Except for a mild degree of intermittent claudication which had been present for 2 or 3 years, the patient was in apparent good health until 2 weeks prior to admission, when he developed coryza, mild malaise and generalized aching, with

TABLE 2. *Laboratory Data in Case 1.*

	Blood								Spinal Fluid		
	Red-cell count	Hemoglobin	White-cell count	Differential count				Heterophil test	Cells	Total protein	Hetero-phil test
Date				Neutro-phils	Lympho-cytes	Mono-cytes	Eosino-phils				
	$\times 10^6$	gm./100 cc.	$\times 10^3$	%	%	%	%	serum dil.	per cu.mm.	mg./100 cc.	
Aug. 26	4.91	14.7	6.2	72	22	5	1	1:56			
Aug. 29			11.6	25	69	5	1				
Aug. 30			15.1	20	76	2	2				
Aug. 31								1:112			
Sept. 4									4	91	
Sept. 5			17.8	19	76	5	0	1:112			
Sept. 6									3		Neg.
Sept. 14	4.80	15.6	10.2	20	72	7	1	1:896			
Sept. 26			9.9	40	54	3	3	1:7			
Oct. 7			9.5	30	64	2	1				
Nov. 16									0	44	
Nov. 17	4.76	14.5	8.0	41	58	0	1				

the face; complete bilateral facial paralysis; bilateral weakness of the trapezius muscle; generalized motor weakness throughout the body, slightly greater on the right side than on the left; generalized contraction of the visual fields, somewhat more marked on the left than on the right; and athetoid movements of the hands and fingers.

Electroencephalograms were made on November 20 and December 1. These were interpreted as mildly abnormal tracings consistent with diffuse encephalopathy.

Despite prolonged hospitalization, physiotherapy, abundant vitamin therapy and a nutritious diet, no additional improvement was obtained, and the patient was discharged from the Army on December 27.

In March, 1945, about 200 days after the onset of his illness, the patient reported that his speech was somewhat better

some tingling in the hands, arms and face. This group of symptoms persisted without appreciable change until admission.

Physical examination revealed a well developed and well nourished Negro in no apparent discomfort. The temperature was 98°F., the pulse 88, and the respirations 16. The blood pressure was 114/76. Numerous small oval patches of dark-brown pigmentation were scattered irregularly over the trunk and extremities. Peripheral vessels were compressible but easily rolled beneath the fingers. There was moderate enlargement of the anterior and posterior cervical and axillary lymph nodes bilaterally and slight enlargement of the inguinal nodes bilaterally, with no tenderness of any of the nodes. There was bilateral pes planus. No note was made of the reflexes. The laboratory findings are shown in Table 3.

TABLE 3. *Laboratory Data in Case 2.*

	Blood								Spinal Fluid	
	Red-cell count	Hemoglobin	White-cell count	Differential count				Heterophil test	Cells	Total protein
Date				Neutro-phils	Lympho-cytes	Mono-cytes	Eosino-phils			
	$\times 10^6$	gm./100 cc.	$\times 10^3$	%	%	%	%	serum dil.	per cu.mm.	mg./100 cc.
Nov. 24	4.60	14.4	4.4	58	40	2	0			
Nov. 27	4.68	14.4	7.9	54	41	5	0			
Dec. 1	4.51	14.4	6.0	58	36	4	2	1:56		
Dec. 4									1	48
Dec. 6			6.5	60	30	8	2	1:112		
Dec. 9	4.57	14.4	6.1	63	30	7	0	1:56		
Dec. 13									2	44
Dec. 19			5.9	45	55	0	0	1:56		
Dec. 26			4.1	36	60	4	0			
Dec. 29			4.6	63	33	4	0			
Jan. 9			4.8	62	27	8	3			
Jan. 30	4.19	13.5	11.7	73	16	7	4			
Feb. 12	3.91	12.9	4.2	65	27	6	2	Neg.		
Feb. 21	4.70	14.7	4.9	52	40	6	2			
Mar. 29	4.75	15.0	5.1	52	48	0	0			

and that he could smile slightly, but that he still had loss of taste, numbness of the face and inability to look upward and had developed a high-pitched whistle in the right ear.

CASE 2. A 36-year-old Negro entered the hospital on November 28, 1944, complaining of frontal headache, malaise and generalized aching. The family history was not remarkable except that a maternal uncle had died of hypertensive heart disease. The past history included measles, chicken pox and whooping cough in childhood, without complications or sequelae, uncomplicated mumps in 1926, malaria in 1923, with several recurrences, the last being in 1927, and tonsillectomy in 1924. There was no history of head trauma or

The blood Kahn reaction was negative, as were x-ray studies of the skull, sinuses and chest.

On bed rest the patient became more comfortable, but on December 1 the frontal headache became severe and he complained of numbness of the chin. On the same day he noticed some difficulty in chewing, could not control the movements of his tongue and developed weakness of the arms and legs. These symptoms continued, with accentuation of the headache at night, so that by December 4 the following physical findings were apparent: a completely expressionless face with inability to smile, whistle, frown or close the eyes completely; Bell's phenomenon, in which the eyes roll upward when the patient tries to close his eyes; slight ptosis of the eyelids; hyperesthesia over the temporomandibular region;

anesthesia of the chin from the mental foramen of one side to that on the opposite side; dysarthric, slurred speech; absence of the gag reflex; paresis of the masseters and pterygoids; diminished muscle reflexes in the upper extremities; absent knee jerks and ankle jerks; diminished superficial abdominal and cremasteric reflexes; a normal plantar reflex; and adiadokokinesis.

The temperature was normal except on December 1 and 2, when it reached 100 and 99.6°F., respectively. During the next few days the gag reflex returned, and enough strength returned to the masseters so that the patient could eat semisolid food. The frontal headache persisted, being mild during the day but severe at night. Considerable tenderness of the facial muscles was also noted during this phase. The generalized lymphadenopathy was considerably reduced by December 10 and became unnoticeable by December 14.

On December 15, the patient complained of increased aching of the muscles of all extremities. Examination at that time showed generalized muscle tenderness and weakness, with marked weakness of the biceps, triceps, thigh extensors and hamstring muscles, the paresis being somewhat more pronounced on the right than on the left. No further change was noted until December 26, by which time the headaches had ceased. By January 3, 1945, the patient had recovered sufficient strength in the right upper extremity to write a short letter. This was the first objective evidence of improvement.

On January 4, a right-sided foot drop was observed; this had probably been present previously but had not been noticed until the patient was allowed out of bed. On the following day some improvement in the muscle strength of the extremities was apparent, the muscle soreness becoming less as the tone improved. From this date onward the patient made gradual, albeit limited, improvement. By January 15 he had regained some use of the facial muscles, being able to smile slightly and raise his eyebrows. The chin anesthesia was also diminished by this time, and the speech was better, although still somewhat thick. The gait was of the waddling and shuffling type, with a right-sided foot drop. On January 18, the superficial abdominal reflexes were found to be brisk. The cremasteric reflexes wers still abnormal, however, in that stimulation of the medial surface of the thigh resulted in lower abdominal reflex spasm but caused no contraction of the dartos muscle. Graduated physiotherapy was begun on January 19, and subsequent improvement was gratifying, the speech becoming practically normal by January 23, the facial expression returning in some measure and the foot drop becoming unnoticeable by January 27. Ophthalmologic examination was negative except for a slight contraction of both visual fields, which remained stationary. Further progress was slow and of limited extent. Except for the first few days of hospitalization, when the patient was drowsy and apathetic, he was mentally alert and cheerful throughout his illness.

General examination on March 17 showed some residual facial weakness, with inability to frown or whistle, and moderate weakness of the pterygoids and temporal muscles. The sternocleidomastoid muscles and those of the extremities were generally soft and flabby, with poor tone and strength; the reflexes in the upper extremities were hypoactive. Knee jerks and ankle jerks were absent, and there was a waddling gait. Finger-to-finger and heel-to-heel tests were well performed.

Physical examination on April 6 showed slight improvement in muscle strength of the extremities and sternocleidomastoids, but the thigh and calf muscles were still tender on deep pressure and squeezing. Stiffness of the upper lip was still apparent on talking and in drinking from a glass, and the gait was shuffling. There was a fine tremor of the fingers and some tremor of the arms and legs when placed on tension. The muscle reflexes of the upper extremities were quite hypoactive; the knee jerks and ankle jerks were absent, but the superficial abdominal and cremasteric reflexes had become active and normal. At that time, 5 months after the onset of illness, it was believed that complete recovery was unlikely and the patient was discharged from the Army on April 14.

Discussion

At the time of admission of both these patients the clinical impression was that of infectious mononucleosis. When the paralytic symptoms appeared, the differential diagnosis broadened. One of the diseases considered was poliomyelitis. No report on sheep-cell agglutination tests in the acute stages of this disease is to be found in the literature, but Hammon and Lawson[34] at the University of California performed Paul–Bunnell tests on serums from 13 patients with paralysis due to poliomyelitis and found that no titer was higher than 1:28.

Clinical observations of these cases support the concept of infectious mononucleosis as a generalized infection with specific localization in one or more of the tissues or organs of the body.[29] In the cases described above there was apparently diffuse and spotty involvement of the nervous system. The clinical picture is similar to that produced by viruses of other diseases and suggests that this too may be a virus infection.

The recognition of disease of the central nervous system due to infectious mononucleosis is a simple matter if one is acquainted with the knowledge that it occurs. The actual incidence of such complications is difficult to estimate and is probably quite low. The important thing, however, is the realization that infectious mononucleosis is not always a benign disease. Patients with this disease should not be dismissed lightly, but deserve careful medical observation and attention.

Summary

A review of the literature indicates that infrequent cases of infectious mononucleosis causing disturbances in the central nervous system have been observed.

Cases of fatal respiratory paralysis due to this disease have been recorded.

Two cases are reported in which infectious mononucleosis resulted in such severe damage to the central nervous system that the patients were declared unfit for further military service.

It is emphasized that infectious mononucleosis is a potentially serious disease, worthy of respect as a generalized virus infection that may threaten life.

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MEDICAL PROGRESS

FLUORINE AND DENTAL CARIES

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PREVALENCE OF DENTAL CARIES

DENTAL caries is perhaps the most widespread of all diseases of mankind and is one from which very few persons escape, especially in civilized races. It has been demonstrated that a positive correlation exists between the primitive (carnivorous) diets of Eskimos in Alaska and their relative freedom from dental decay.[1] A high prevalence of dental decay, however, is found among Eskimos who live in cities and consume a civilized diet. Practically every adult and almost every child over six years of age has suffered from dental defects.[2] These defects often induce a train of related maladies that could have been prevented by proper dental care. Dental diseases are not limited to any group and attack all persons, regardless of age, sex, geographical location or economic status.

Dental caries is the destroyer of most teeth that are lost by persons under thirty; thereafter pyorrhea assumes first rank as the cause of loss of teeth. Caries begins as soon as a child has teeth. Over ninety-five per cent of children ten years of age have one or more decayed permanent teeth. By the time the child reaches eighteen, nine permanent teeth, on an average, have become decayed and several extractions have had to be made. The loss of certain teeth upsets the proper alignment of others and brings about malocclusion and predisposes to pyorrhea, the prime cause of loss of teeth in later life. Moreover, diseased teeth and gums may cause disease, directly or indirectly, in adjacent as well as other parts of the body.

Once a defect is produced by decay, evidence of its existence is never obliterated.[3] It is therefore possible by direct examination to determine what has happened to teeth in the past. The permanent teeth with defects caused by present and past attacks of caries are classified in three groups — decayed teeth, those with open carious lesions requiring treatment by fillings; missing teeth, those already extracted or requiring immediate extraction because of extensive destruction of caries; and filled teeth, those previously carious but with evidence of treatment in the form of fillings.[4] By common usage and for the purpose of brevity, teeth so affected are collectively designated by the symbol "DMF" (decayed, missing and filled).

Because of the characteristic accumulation of caries stigmas with increasing age, the increment in the number of DMF teeth or of DMF surfaces per year per person may be taken as a measure of the incidence of caries. The prevalence of caries may be measured in several ways — by the ratio of the number of persons per 100 population having one or more DMF teeth; the total DMF teeth per person; and the number of DMF surfaces per person, which gives a fuller comparison than either of the foregoing, since each tooth may be attacked on several surfaces.

EPIDEMIOLOGY OF DENTAL CARIES

Age

One of the most significant factors associated with increased dental caries is age. Studies made in 1940 by Klein and Palmer[5] on 6200 children in Hagerstown, Maryland, and in the same year by Sloman and Sharp[6] on 6300 high-school children in San Francisco yielded definite evidence of the accumulative destruction caused by dental caries. At six

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years of age, 15.9 per cent of the Hagerstown children had one or more DMF teeth; the percentage increased each year until by eighteen years of age it was 96.6 per cent. The San Francisco high-school children showed an increase from 91.0 per cent at the age of thirteen to 97.2 per cent at eighteen. If the second method of analysis is used, the number of DMF teeth per child in the Hagerstown group increased from 0.29 at the age of six to 9.25 at nineteen, and that in the San Francisco children increased from 3.8 at the age of twelve to 8.3 at eighteen. When the same method of comparison was applied to an adult population in New York City in 1939 by Hollander and Dunning,[7] it revealed a steady increment of tooth destruction with advancement in age; for example, at twenty to twenty-four years there were 15.82 DMF teeth per person; at forty to forty-four years, 21.61; at fifty to fifty-four years, 22.36; and at sixty to sixty-four years, 23.37. If the third method of comparison (DMF surfaces per person) is applied to the Hagerstown and New York City data, it is found that there was a constant increase of tooth decay with advancing age. At six years of age the Hagerstown children averaged 0.38 DMF surfaces per child, at ten years 4.87 and at fifteen years 13.35, whereas in adults in New York City the average increased from 31.52 at the age of twenty to twenty-four to 70.09 at the age of sixty-five and over.

Sex

Several observers[8–11] have noted a greater prevalence of dental caries among females than among males, but this is probably due to the earlier eruption of the permanent teeth rather than to a predisposition to the formation of caries. Females have a greater amount of dental decay than do males of the same age. In 1938, however, Klein and Palmer[12, 13] demonstrated that this difference is also due to the fact that permanent teeth erupt earlier in girls than they do in boys. Actually, therefore, if a comparison of the age of the teeth is made for the two sexes, there is no significant difference in dental decay until the age of fifteen years. Thereafter there is a gradual increment of dental decay in females with advancing age.

Race

Only scattered data on the difference in the prevalence of caries observed in different races are available. They indicate that race is a significant factor. Negro children have lower rates of DMF teeth per person than do white children,[3] the difference increasing with age. Thus, at ten years of age, in the white children of Hagerstown and Baltimore[4, 5] the rate was 2.5, compared with 1.6 in Negro children of the same communities. In Indian children of the same age examined by Klein and Palmer[14] the rate was 1.3, whereas in Navajo children of this age it was 0.5. At fifteen years of age the rate for Hagerstown white children was 6.6, that for San Francisco white children was 6.5, that for Hagerstown Negro children was 4.1, and that for Navajo children was 3.1. In other words, the white children had about one and a half times as many decayed, missing or filled permanent teeth as did the Negro children and twice as many as did the Navajo children.

Familial Susceptibility

Several investigators[15–20] have studied familial susceptibility to dental caries. Although their findings indicate a significant difference between different families, it is not clear whether these observed differences are in reality due to inherited (Mendelian) factors or whether they are not more probably due to environmental factors, such as family eating habits, consumption of candy, poor economic status and other factors that are common to the members of the same household.

Klein and Palmer[21] studied 4400 white elementary-school children in Hagerstown and found 117 with extreme susceptibility to caries and 184 with immunity to it. The susceptible children had 182 siblings, whereas the immune children had 306, all of whom had been examined. Studies of these records revealed that the tendency toward caries of the siblings of immune children was about half that of the siblings of susceptible children.

Economic Status

The literature tends to suggest that economic status does not markedly affect the prevalence of caries, although the amount of dental work is related directly to the income of the family.[22–24] Klein and Palmer[25] studied the caries prevalence rates for 200,000 children in forty New Jersey communities. They concluded that the intrinsic tendency of children to experience attacks on the permanent teeth by caries does not depend on the economic status of the community in which they live. The volume of dental care in the form of fillings in the permanent teeth, however, markedly increased with improvement in the economic level of the community; the odontothanatoxic rate (the number of permanent teeth extracted and indicated for extraction per 100 children) diminishes as the economic level rises.

Conclusions contrary to these reports were reached by Hyde,[26] who examined inhabitants of Massachusetts at the Boston Armed Forces Induction Station. The incidence of caries (DMF teeth) per person was highest in the most prosperous communities and lowest in the poorest communities. The English and Irish had the greatest amount of dental decay, whereas the Negroes and Chinese had the least. Three times as many selectees from Irish communities as from Portuguese or Russian (Jewish) communities were rejected for dental defects. The evaluation of the influence of economic factors on

dental caries must in the final analysis await a detailed study of other factors that are usually related to economic ones, such as diet and outdoor activity.

Geographical Location

There appears to be a definite correlation between the prevalence of dental caries and several factors involved in geographical location. These factors pertain not only to certain locations within the United States but also to geologic formations and climatologic differences. Mills,[27] after studying the prevalence of caries in different latitudes, concluded that rates are highest in the northern and lowest in the southern areas of the country. In latitudes 25 to 26.9° the prevalence rates of DMF teeth were under 300 per 100 boys, whereas in latitudes 37 to 48° they were above 300. The rate increased by about 15 units for each degree farther north. East[28] believes that areas having high annual total hours of sunshine have less caries than do areas with fewer hours of sunshine. Nizel and Bibby[29] are convinced that geographic variations have a definite influence on the prevalence of dental caries, as was demonstrated on examination of 22,117 soldiers in Camp Edwards, Massachusetts. Many earlier observers — Lewis[30] in 1865, Britten and Perrott[31] for draftees in World War I, Ferguson[32] for naval recruits and Senn[33] for aviation cadets in World War II — have shown that New Englanders had a higher exemption rate because of dental defects than did draftees from other sections of the country. Nizel and Bibby believe that the consistent repetition of this phenomenon over a period of eighty years eliminates all possibility of chance and establishes geographic variation in the prevalence of dental decay. The geographic prevalence of caries in draftees and recruits is summarized in Table 1.

TABLE 1. *Prevalence of Caries in Draftees and Recruits.*

STATE	DRAFTEES[30] 1863–1864		DRAFTEES[31] 1918		NAVAL RECRUITS[32] 1934		SELECTEES[33] 1943	
	RATE OF REJECTION FOR LOSS OF TEETH	ORDER OF PREVALENCE‡	RATE OF REJECTION FOR DEFECTIVE AND MISSING TEETH	ORDER OF PREVALENCE	AVERAGE NO. OF DECAYED, MISSING AND FILLED TEETH PER RECRUIT	ORDER OF PREVALENCE‡	AVERAGE NO. OF DECAYED AND MISSING TEETH PER SELECTEE	ORDER OF PREVALENCE‡
	per 1000		*per 1000*					
Alabama			9.30	39	3.50	24†	10.07	22
Arizona			7.29	42	6.00	16*	8.87	37
Arkansas			7.90	49	3.00	27	7.76	44
California			14.02	31	6.00	16*	8.93	35
Colorado			10.24	37	6.00	16*	8.32	40
Connecticut	26.53	3	36.52	8	12.54	1	10.97	14
Delaware	7.62	15	35.02	9	9.70	9*	17.05	1
Florida			23.03	15	3.60	23	8.79	39
Georgia			21.19	19	3.40	25†	9.99	26
Idaho			16.58	27	6.00	16*	11.59	11
Illinois			17.48	25	9.25	10	10.12	20
Indiana			9.47	38	4.38	19	10.18	19
Iowa			12.76	35	4.10	21	9.31	29
Kansas			5.89	48	4.00	22*	7.81	43
Kentucky	10.98	13	8.75	41	4.30	20	10.02	25
Louisiana			23.71	13	4.00	22*	9.20	31
Maine	10.21	14	79.45	3	10.20	8*	15.96	2
Maryland	16.57	11	21.28	18	9.70	9*	10.62	15
Massachusetts	34.87	1	78.82	5	12.20	2	12.06	8
Michigan	13.11	12	34.50	10	7.14	13	10.27	18
Minnesota	22.08	7	20.06	21	9.10	11	10.06	23
Mississippi			13.31	33	3.40	25†	8.92	36
Missouri			6.52	44	4.40	18	10.11	21
Montana			23.17	14	6.00	16*	8.84	38
Nebraska			6.13	45	6.80	15	9.14	32
Nevada			13.45	32	6.00	16*	7.90	42
New Hampshire	25.05	5	92.03	2	10.20	8	12.15	7
New Jersey	20.19	9	52.38	6	10.30	7	12.43	6
New Mexico			5.93	46	6.00	16*	7.67	45
New York	27.56	2	43.13	7	10.84	5	13.88	4
North Carolina			17.07	26	3.50	24†	8.95	33
North Dakota			19.02	22	6.00	16*	10.39	17
Ohio			14.62	30	10.53	6	11.70	10
Oklahoma			6.95	43	4.00	22*	7.37	46
Oregon			27.38	12	6.00	16*	12.62	5
Pennsylvania	25.70	4	29.08	11	11.40	3	10.46	16
Rhode Island	22.46	6	79.26	4	11.00	4	15.39	3
South Carolina			18.81	23	4.00	22*	9.23	30
South Dakota			20.91	20	5.30	17	9.64	28
Tennessee			11.04	36	3.20	26	8.95	34
Texas			5.91	47	4.00	22*	6.44	48
Utah			18.71	24	6.00	16*	9.89	27
Vermont	20.57	8	102.85	1	10.20	8	11.00	13
Virginia			21.33	17	4.00	22*	8.31	41
Washington			27.91	16	6.00	16*	11.23	12
West Virginia			13.21	34	7.90	12	10.05	24
Wisconsin	18.87	10	16.03	29	7.00	14	11.98	9
Wyoming			9.28	40	6.00	16*	6.69	47
District of Columbia	5.39	16	16.15	28	9.70	9*		

*Owing to the small number of recruits from certain areas, those from contiguous states were tabulated together and each state was given the same rate.

†Figure happened to be the same for these states.

‡States are arranged in the order of descending prevalence.

The role of the mineral composition of public water supplies in relation to dental caries has been extensively studied by many authors, especially Dean[34] and East.[35] Both positive and negative relations between hardness of water and prevalence of caries have been reported by different investigators. East[36] offers evidence that users of hard water have less caries than do users of soft water. It has, however, been pointed out by Dean[37] and many others that this phenomenon is not the result of hard waters but rather the presence of fluorides in them. The role that fluorine plays in the prevention of caries is discussed in detail later in this paper. Nizel and Bibby, however, noted that caries is prevalent in regions with podzol and gray-brown podzolic soil and that wide variations in susceptibility to it occur in association with the red and yellow, chernozem and chestnut and prairie-soil regions of the south and the midwestern states. Finally, these authors urge more extensive investigation of soils in relation to dental caries, pointing out that there may be other rare elements besides fluorine that influence the incidence of dental decay.

Diet

McCollum,[3] after examining the literature relating to the etiology of dental caries during recent years, concluded that there is an agreement among authors that carious lesions are caused by acid decomposition of the tooth enamel and subsequently the dentin, associated with proleolytic destruction of the organic substance of the tooth. There is unanimity of opinion that caries is restricted to persons and animals that have a liberal carbohydrate diet, whereas carnivorous men and animals do not suffer from this disease. Dental caries does not attack the surfaces of teeth indiscriminately but occurs only at sites that favor the lodgment of food particles or on surfaces of the enamel that harbor mucinous plaques. In these sites, acid is formed by fermentation of carbohydrates by micro-organisms and is protected by the plaques against being washed away by saliva or neutralized by salivary alkalinity.

Faulty structure in the enamel may cause the lodging of food particles in fissures and pits and thereby predispose to the formation of dental caries. The filling of these defects prior to decay is known as prophylactic odontotomy and has proved to be an effective means of prevention of caries. Such defects, however, may be the result of dietary deficiency. When, owing to vitamin A deficiency, the enamel-forming cells are injured, enamel prisms that are less dense than normal ones or are incomplete as to length and imperfectly fitted together form defective enamel. In severe cases vitamin A deficiency causes hypoplastic teeth, characterized by thin enamel deficient in hardness; in milder deficiency there may be pitting.

Vitamin D deficiency, with the disturbance of calcium and phosphate metabolism of rickets, may cause defects in teeth, since the enamel-forming cells are unable to withdraw from the blood the materials required for the formation of normal cells. McBeath[38] and Erpf[39] found a seasonal variation in the incidence of dental caries; the highest incidence of new cavities occurred in late winter and spring, when the amount of sunshine was the lowest. It is thus apparent that the lack of sunshine may be to blame for a lowered production of vitamin D in the bodies of the children as a result of the reduced amount of ultraviolet light. This decrease was manifested in part by the increased amount of dental caries observed during the period of low vitamin D production.

The eating of sugar and candy has been believed by many authors to cause dental decay. Bunting and his associates[41] were able to demonstrate that children classed as immune to caries who were allowed to eat about 3 pounds of candy a week soon became susceptible to dental decay. Thus, sugar seems to be a greater menace to the teeth than are starchy foods. This observation has been verified by studying the diets of starch-eating primitives, who usually have relatively small amounts of dental decay.

In conclusion, it may be said that if a person has an optimal diet during the developmental period of the teeth, they will be free from defects such as pits, fissures and hypoplastic enamel. Subsistence throughout life on a strictly carnivorous diet prevents caries. The presence of considerable carbohydrate, especially sugar, in the diet is likely to predispose to dental decay. The elimination of sugar from the diet, with the substitution of starch, affords a less favorable oral medium for the development of acid-forming organisms and so protects the teeth against acid decomposition. Vigorous chewing of raw fruits or raw vegetables, which also act as detergents, tends to prevent dental caries.

Mouth Bacteria

Bibby[3] summarized as follows the reasons why bacteria are believed to be of fundamental importance in causing dental caries: tooth destruction progresses from the outside of the teeth inward; the sites at which caries occur are the places at which foods accumulate; an association has been shown to exist between the acidogenic properties — carbohydrate content — of diets and their effects in producing caries; as yet acids are the only chemical and biologic agents that are known to have the power of destroying dental enamel and bringing about decalcification of dentin; and lastly, bacteria are the only agents existing in the mouth that are known to have the power of producing acid from foodstuffs.

Accumulated evidence indicates that acid is definitely concerned in tooth decay. Enright et al.[40] demonstrated that cavities like those of caries can be produced by the action of acids. Bunting[41] and

Bibby[42] were able to produce similar cavities by bacteria grown in the presence of carbohydrates. Fosdick[43] found that the acidity of dental mucous plaques is increased when fermentable carbohydrates are present. Deakins[44] demonstrated that the chemical changes that occur during the formation of caries correspond to those caused by the action of acid. Miller[45] and Kanthak[46] found organic acids in carious dentin. Finally, Miller[47] and Hodge and Finn[48] were able to demonstrate that certain agents that prevent acid formation from carbohydrates prevent dental caries in animals, and Dean, Jay, Arnold, McClure and Elvove[49] and Day[50] were able to demonstrate the same fact for man.

Culture studies by various investigators have led them to postulate a causative relation between dental caries and certain bacteria; on this basis lactobacilli, streptococci, fusiform organisms, species of Cladothrix and Leptothrix and anaerobic bacteria have been named as etiologic agents of dental caries. Bunting et al.[51] have reported an extensive study of the association between increases in the number of lactobacilli and dental caries; lactobacilli were shown to decrease when cavities were filled or carious teeth were extracted. As yet no specific bacteria have been positively proved to be the prime cause of dental decay. It may therefore be concluded that several types of bacteria are associated with dental decay and that any organism that produces acid in the mouth is a potential cause of decalcification. Experiments conducted by Bibby,[42] however, indicate that lactobacilli are present in a relatively small number and may be caused by conditions brought about through dental caries, whereas streptococci are present in relatively large numbers, grow more rapidly, are active in bringing about chemical changes and may in effect be the bacterial flora chiefly responsible for dental decay.

Cost of Dental Care

It is readily understood that the repair of the constant damage produced by dental caries is costing the Nation large sums of money annually. These costs are for repairing teeth that develop cavities, extracting those that are beyond repair and providing replacements for lost teeth that are required for the mastication of food. Detailed information regarding the prevalence of dental defects among the entire adult population of the United States is limited. Surgeon General Parran[2] in a statement before a congressional committee in a hearing on a bill to provide greater facilities for dental care gave an estimate, prepared by one of the dentists of the United States Public Health Service, of the accumulated dental needs of the population. In the same statement he gave a list of the average charges for various items in dental care. On the basis of these figures Table 2 has been prepared. It shows that it would cost over $4,000,000,000 to repair the damage done by dental caries and pyorrhea to the teeth of the American population, the share of Massachusetts being nearly $134,000,000. This amount represents a per capita cost of $31.00. Once the teeth were put in good repair, the constant inroads of dental caries would produce additional damage, the repair of which would cost further sums.

It is obvious that there are not sufficient dentists and dental assistants in the Nation to take care of this accumulated volume of dental work. The best study available on the total annual expenditures for dental care in the United States is that made in 1929 by Leven and Beck,[52] who estimated the gross annual income of dentists as $446,000,000, about one ninth of the $4,000,000,000 mentioned above. In other words, it would take the men now trained in dentistry approximately nine years to care for the accumulated needs of the American people.

TABLE 2. *Dental Needs for the United States.*

ITEMS	NO. NEEDED	AVERAGE COST PER ITEM	TOTAL COST	COST FOR MASSACHUSETTS
Extractions	238,500,000	$2.00	$477,000,000	$15,700,000
Fillings	632,000,000	3.50	2,212,000,000	72,600,000
Prophylaxes	125,000,000	2.00	250,000,000	8,200,000
Dental treatments	20,000,000	2.00	40,000,000	1,300,000
Crowns and bridges	39,500,000	15.00	592,500,000	19,500,000
Partial dentures	20,000,000	25.00	500,000,000	16,500,000
Totals			$4,071,500,000	$133,800,000

On the premise that the destruction of teeth by dental caries and pyorrhea will continue at the present rate, Dollar[53] has estimated the annual cost of maintaining the teeth of this population in good repair as $1,300,000,000. This is about three times as much as is being expended for dental service at the present time. The figure for Massachusetts would be almost $43,000,000.

It is self-evident that if the inhabitants of this country are to have good teeth, either there must be provided a sufficient number of dentists and dental assistants who are adequately paid or ways must be found for halting the inroads of dental caries and pyorrhea. This is the reason why those interested in dental health[37, 54, 55] are looking so hopefully toward the use of fluorine and of other agents that give promise of reducing the annual damage by diseases of the teeth and gums. For this reason also, health departments should have a part in carrying on studies that will uncover other measures for stopping this annual drain on the health, energy and financial resources of the Nation.

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 32231

PRESENTATION OF CASE

A fifty-one-year-old housewife entered the hospital because of burning, tingling and pain in the fingers.

The patient was allegedly well until six months before admission, when she began having a dull aching in her fingertips followed several weeks later by severe burning and tingling. At first these symptoms came in paroxysms, but they gradually became constant, lasting day and night and spreading to involve the entire hands. There was no definite story of weakness, but the paresthesias limited the use of her hands to such an extent that they became stiff and she was finally unable to feed herself. She saw her physician, who diagnosed anemia and treated her with liver and iron injections, as well as with many vitamin preparations. During this medication she developed anorexia, and three weeks before entry she began to vomit everything she ate. Concomitantly, she noticed swelling of the ankles, mild pain in the calves and some difficulty in walking. The pains in her arms were somewhat less when she sat on the edge of her bed, and she spent much time in that position.

In the past her health had been good except for an attack of gout in the great toe thirty years previously and a tubal pregnancy followed by a salpingo-oophorectomy seventeen years before entry. She had had two normal deliveries. The menopause had occurred four and a half years before entry.

Physical examination revealed a well developed but rather obese pale woman in no acute distress. The mucous membranes were pale. The skin was dry. The tongue had smooth edges, but it was not red and there was no cheilosis. An inconstant Grade I systolic murmur was heard over the apex. The lungs were clear. The abdomen was obese. The arms showed moderate weakness of all motions and marked weakness of the hands. There was considerable limitation of passive motion of the fingers, elbows and shoulders. The wrists and shoulders were somewhat tender. There was hyperesthesia to pain, and vibration and position senses were normal. The thenar eminences were flattened. The legs showed no limitation of motion and no paresthesias. Vibration and position senses were moderately decreased. There was moderate calf tenderness.

The temperature, pulse and respirations were normal. The blood pressure was 150 systolic, 85 diastolic.

Examination of the blood showed a red-cell count of 3,100,000, with a hemoglobin of 9 gm. The smear was reported as normocytic and normochromic. The white-cell count was 23,500, with 90 per cent neutrophils and 9 per cent lymphocytes. The nonprotein nitrogen was 92 mg. per 100 cc., and the protein 5.7 gm. The Hinton and Wassermann tests were negative. The urine showed many white cells in clumps but no albumin or casts. The specific gravity was 1.010. Culture revealed a moderate number of colon bacilli.

X-ray films of the chest and a gastrointestinal series were reported normal.

Lumbar puncture showed an initial pressure equivalent to 170 mm. of water and a final pressure of 140 mm., with free dynamics. There were no cells, and the ammonium sulfate ring-test was negative. The total protein was 21 mg. per 100 cc. Gold-sol and Wassermann tests were negative.

The patient was extremely unco-operative and resisted efforts to mobilize her. She complained constantly of the pains in her joints and muscles. She was oriented during the day, but at night she had severe delirium with nightmares and hallucinations despite sodium amytal sedation.

On the fifth hospital day she was transfused with 500 cc. of whole blood. On the ninth hospital day the white-cell count reached 30,000, with 82 per cent neutrophils and 18 per cent lymphocytes, some of which appeared atypical. Further urine studies showed a + test for albumin, failure to concentrate to a specific gravity of more than 1.014, and less than 5 per cent excretion of phenolsulfonephthalein at the end of two hours. The patient gradually became oliguric and passed into coma. On the nineteenth hospital day the nonprotein nitrogen reached 140 mg. per 100 cc. and the carbon dioxide 7.5 milliequiv. per liter. She died on the twentieth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. MARIAN W. ROPES: I am sure that the one safe statement is that I am unable to make a diagnosis that will completely fit this picture. There are a few obvious facts: the patient was in uremia when she came to the hospital; she had an infection of the urinary tract; she had anemia, leukocytosis and neurologic and skeletal symptoms; and she died of renal failure. Several diagnoses are suggested by these symptoms, and the problem is to pick one that will correlate all the findings.

In any patient with renal disease, anemia and skeletal pain one thinks of multiple myeloma. There is no more than that to support this diagnosis, how-

ever, unless we assume the unlikely possibility that the atypical lymphocytes might have been plasma cells. Such a diagnosis would not explain the degree of muscle involvement or paresthesias. Similarly, with paresthesias, anemia and smooth tongue edges, one thinks of pernicious anemia; but we are told that on at least one examination the blood was normocytic and normochromic. She had had some treatment that might have modified the picture, but there was no evidence in the blood examination that she had pernicious anemia. Furthermore, the neurologic examination was essentially normal. The vibration and position senses were normal, except for slight reduction in the legs. Also this diagnosis would not explain the renal involvement or the degree of muscle involvement. The neurologic findings suggest polyneuritis, but the course is against such a diagnosis. The relatively normal neurologic examination is against it, and particularly against it is the normal spinal fluid. If such a diagnosis were to be made it would be separate from the renal involvement. I think we can rule that diagnosis out. One can also rule out muscular dystrophy. In fact the degree of muscle change is so slight that I doubt that such a diagnosis could even be considered. There was weakness but relatively little, — more pain than weakness, — and the paresthesias were out of proportion for such a diagnosis. There is minimal evidence to suggest leukemia or lymphoma, and I think that they can be ruled out.

In any constitutional disease that has joint and muscle involvement, paresthesias and anemia, we have to think of connective-tissue diseases, such as rheumatoid arthritis, disseminated lupus erythematosus, periarteritis nodosa, dermatomyositis and scleroderma. The last can be easily ruled out because there was no skin involvement. Dermatomyositis is possible because of the degree of involvement of the muscles. Absence of real inflammation of the muscles and absence of weakness in the trunk or pharyngeal muscles would be unlikely in a severe case of dermatomyositis. Again, this would not explain the renal involvement. The other three conditions cannot be ruled out, although I find little evidence for any of them. The joint and muscle symptoms are consistent with rheumatoid arthritis. Unfortunately we have no x-ray films of the joints, and I judge that the other films were negative.

DR. TRACY B. MALLORY: There is one shoulder joint showing in this chest film.

DR. ROPES: In it there is nothing except the suggestion of some loss of calcium from the bones. There is no definite soft-tissue involvement of which I can be sure.

There is a story of a joint attack in the past that was called "gout." I think that this is of no significance so far as the present illness goes. Indeed, I doubt that she had gout. It is extremely rare in women, and an attack of acute arthritis involving the great toe is often called gout without further evidence. My feeling is that she had another type of arthritis in the great toe. We cannot rule it out, but I think that she did not have gout with a progressive arteriosclerosis causing the renal failure.

If rheumatoid arthritis is to be considered as the cause of the recent condition, it would not explain the renal involvement unless the patient had amyloidosis. The development of amyloid in rheumatoid arthritis within a six-month period is unusual. It occurs after various changes in the blood have taken place, such as an increased globulin, but in our experience it does not occur until after at least six months and usually after a year of disease. The evidence we have is that her illness did not start until six months before admission. I doubt that she had amyloid due to rheumatoid arthritis.

Joint and muscle symptoms can occur in disseminated lupus, but here, as in rheumatoid arthritis, one would not expect such severe paresthesias or marked muscle changes. The diagnosis of lupus would, of course, explain the renal involvement and renal failure as the cause of death, but it is impossible to make the diagnosis of disseminated lupus in the absence of involvement of the skin and serous membranes. Furthermore, a leukocytosis of this degree would be extremely unusual, if not unknown, in lupus without obvious infection. In fact the majority of cases of disseminated lupus, even with infection, do not elevate the neutrophil count to this level.

Periarteritis nodosa is perhaps more difficult to rule out despite the fact that there is again no definite evidence for it. The degree of paresthesia is greater than one ordinarily sees with periarteritis nodosa, and the degree of muscle weakness and muscle change is much greater than one ordinarily sees. If the renal involvement were due to periarteritis nodosa one would expect more abnormalities in the urine than a + test for albumin with, on one occasion, many white cells. I am assuming that the other urinary specimens were essentially negative. So the group of diseases suggested by the skeletal and neurologic symptoms do not explain the entire picture.

I might turn to the renal involvement and consider the likely causes of renal failure that lead to death. There are few conditions that show an essentially negative urine at the time of renal failure. From the whole picture — the age, the lack of arteriosclerosis elsewhere and the relatively negative urinary findings — it is unlikely that the patient had nephritis on the basis of arteriosclerotic changes. Chronic glomerulonephritis cannot be ruled out. She went slowly into uremia, and at that time she was known to have an infection of the urinary tract, but again, one would surely expect more changes in the urine. Similarly pyelonephritis cannot be ruled out, although one would expect more changes

in the urine. The strongest point in favor of pyelonephritis is the presence of urinary-tract infection at the time of admission. Unfortunately, we know nothing about the duration of the infection. Apparently there were no symptoms associated with the genitourinary tract before her present illness. In fact the only renal disease that I was able to think of that gives as few changes in the urine is amyloid disease, which frequently shows only albumin in the urine. I have never happened to hear of or see a patient with so little albumin in the urine at the time of death from amyloid disease of the kidney. That would not rule it out, however. We have seen patients who died of amyloid disease who had had nothing except albumin in the urine throughout the course of the disease.

It is of interest to compare this case with one reported by Lengh* in 1937. The patient was a forty-seven-year-old woman who six months before entry had pain in the joints, paresthesias and anemia and at the time of admission had some albumin in the urine. She died soon after her hospital admission and at autopsy was found to have pyelonephritis, with stones and atypical amyloid disease, masses of amyloid being present in the joint tissues. It also involved the tongue and many vessel walls — a few in the liver, spleen and heart and many in the kidneys. The explanation that Lengh gave in this case was that it was amyloid disease secondary to severe infection — a pyelonephritis of years' duration. The involvement of tissues in that case is much like that reported in atypical amyloid disease or so-called "paramyeloidosis," in which the liver and spleen are not involved but amyloid is present in muscles, joints, bones, the tongue and thyroid gland and the vessel walls, often in the kidneys. Interestingly enough this particular atypical form is usually not secondary to any known cause. Infection is generally not found. In the secondary type of amyloidosis in which the liver and spleen are often involved the cause of the amyloid is usually an infection.

In trying to put together the diseases suggested by the neurologic and skeletal symptoms and the diseases suggested by the kidney disease, I find it difficult to offer an adequate explanation of the entire picture. I shall say first that she had an infection of the urinary tract. It is obvious that she had uremia, which was probably due, I shall say, to amyloid disease. Whether or not the amyloid disease was secondary either to one of the group of connective-tissue diseases that I have discussed or to the renal infection, or whether it represented the so-called "atypical amyloid," which would explain all the other symptoms, I cannot tell. The possibility of explaining it on the basis of atypical amyloidosis is intriguing.

*Lengh, F. Zur Kenntnis der Amyloidablagerung in den Gelenken. *Zentralbl. f. allg. Path. u. path. Anat.* 69:1-5, 1937.

Dr. Bernard Jacobson: In addition to amyloid disease, would you not consider Bence–Jones proteinuria as a possibility?

Dr. Ropes: I mentioned Bence–Jones proteinuria as a cause of the renal disease, but in the absence of other evidence I ruled it out. The renal evidence is satisfactory, but I cannot explain the rest of the picture on that basis.

Dr. Chester M. Jones: We did not know what the patient had. We thought of many of the things that Dr. Ropes has outlined. As a matter of fact, chronic renal disease was not too seriously considered for several days because the first and second specimens of urine had no albumin; the third and fourth specimens gave only a + test for albumin and contained white cells but no other formed elements. My feeling was that she had chronic glomerulonephritis. She had a fixation of gravity at 1.010, and practically no dye was excreted in the phenolsulfonephthalein test. The other findings were those of a person who had prolonged anorexia and vomiting, with malnutrition and deficiency disease, and possibly a thiamin lack, with a peripheral neuritis, for which she was also treated. Incidentally she had been treated with liver by her own doctor without any apparent improvement. Dermatomyositis was considered, but there was no evidence that that existed.

Clinical Diagnoses

Chronic glomerulonephritis.
Uremia.
Peripheral neuritis.

Dr. Ropes's Diagnoses

Chronic pyelonephritis.
Uremia.
Amyloid disease (? atypical form).

Anatomical Diagnoses

Diffuse plasma-cell myeloma.
Lower nephron nephrosis (Bence–Jones proteinuria type).
Atypical or "primary" amyloid disease, involving muscles, joints, heart and blood vessels.
Chronic pyelonephritis, minimal.

Pathological Discussion

Dr. Mallory: Obviously this was a very difficult case for diagnosis, and Dr. Ropes has come astoundingly close. Everything can be explained on the basis of one condition and its recognized complications. The patient did have extensive amyloid disease with the distribution of the so-called "atypical" or "primary" type, with marked involvement of the muscles, joints, heart and blood vessels. The liver, spleen and kidneys were entirely free from the disease. The kidneys showed massive plugging of all the lower nephrons with hyaline casts and severe interstitial nephritis, with a few foci of pyelonephritis, which were relatively unimportant.

One very peculiar and I think probably pathognomonic finding was that there were foreign-body giant cells around the casts within the tubules. I have never seen that except in Bence–Jones proteinuria. I was first shown the kidney in this case, and because of this finding asked to see the bone marrow, which proved to be diffusely invaded with plasma cells. So the primary diagnosis is diffuse plasma-cell myeloma, with Bence–Jones proteinuria, the so-called "myeloma kidney." Amyloid disease is a recognized sequel of myeloma and oddly enough nearly always shows the distribution of so-called "primary" amyloidosis rather than that of the usual secondary type.

DR. JACOBSON: What was the serum protein value?

DR. JONES: It was 5.7 gm. per 100 cc. The service considered doing a bone-marrow biopsy, but the patient was so ill that it was not done.

DR. MALLORY: I do not believe that the serum protein value was checked.

DR. ROPES: The low serum protein might be explained by the fact that she had lost albumin in the urine over a long period.

DR. JACOBSON: It is interesting that this is one of the few cases autopsied here that has shown atypical amyloid distribution.

DR. MALLORY: I can remember only one other.

CASE 32232

PRESENTATION OF CASE

A fifty-three-year-old shoe cutter entered the hospital because of progressive weakness and loss of weight.

The patient had been in apparent good health until ten months before entry, when he vomited four times in one night. He went to a local hospital, where x-ray films were taken, but was sent home with some pills. He did not vomit again, but became progressively weaker and lost about 15 pounds. Several weeks before entry he noticed increasing anorexia, constipation, flatulence and a feeling of abdominal fullness. The stools were darker than usual, but not black, and he noticed no blood. He also suffered from sour stomach, which was aggravated when he was lying down.

Physical examination showed a pale, lethargic man, with evidence of recent weight loss. The mouth was edentulous. The heart and lungs were normal. At the level of the umbilicus there was an irregular, hard, freely movable abdominal mass, about 12 cm. in diameter, which could readily be shifted to either side of the umbilicus and caused marked local protuberance of the abdomen. Peristaltic sounds were rather high pitched. The liver could not be palpated. Rectal examination was negative except for moderate enlargement of the prostate.

The temperature, pulse and respirations were normal. The blood pressure was 120 systolic, 60 diastolic.

Examination of the blood revealed a hemoglobin of 8 gm. The white-cell count was 8200. The nonprotein nitrogen was 31 mg. and the total protein 6.8 gm. per 100 cc., and the chloride 102 milliequiv. per liter. The prothrombin time was normal. The urine was normal. Repeated stool examinations were guaiac positive.

X-ray examination of the abdomen, including an intravenous pyelogram, showed normal kidneys. There were several diverticulums of the urinary bladder, measuring up to 4 by 1.5 cm. A barium enema readily filled the entire colon and terminal ileum, showing them to be normal in form and function. The colon contained some fecal material, particularly in the ascending and proximal transverse portions. On the post-evacuation film the proximal transverse colon was displaced slightly upward by extrinsic pressure from the midabdominal mass, which was shifted upward as compared with its position on the pre-evacuation film (Fig. 1). A small intestinal enema revealed an area of ulceration, measuring approximately 6 cm., in the upper jejunum, within the freely movable palpable mass (Fig. 2). The bowel was slightly narrowed in the region of the ulceration, but most of the mass lay outside the bowel lumen, pushing aside the remainder of the intestinal loops in this area. Some of these loops seemed to be attached to the tumor mass.

An operation was performed on the ninth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. HORACE K. SOWLES: May we see the x-ray films?

DR. RICHARD SCHATZKI: The mass is fairly well visualized in the post-evacuation film, where it displaces the colon (Fig. 1). The films of the small intestine again outline the mass that displaces the loops of intestine, but one is seen going straight through the mass, where the ulceration described in the report (Fig. 2) is visible. It can be seen even better on the spot films, which also show slight dilatation of the jejunum proximal to the narrow ulcerative region.

DR. SOWLES: This patient had symptoms of intestinal obstruction, although he had only one real attack. On admission, he had a low hemoglobin, a history of loss of weight and weakness. One immediately thinks of neoplasm of the right colon, in which the only presenting symptoms are sometimes anemia and loss of weight and weakness. As we go along in the history we find that the colon apparently was normal by barium enema whereas the small bowel showed a definite tumor in the jejunum — a large tumor with ulcerations of the mucous membrane. I think we can rule out carcinoma, which is quite rare in the jejunum, and does not

present a tumor of this size. It is usually a small tumor, whose only symptom is intermittent obstruction, and that does not fit the picture in this case, because this is a large tumor with extensive ulceration of the mucous membrane as shown by x-ray and by the occult blood in the stools.

The time is nearly up and I shall not discuss this any further. I shall say that the most probable diagnosis is tumor of the small bowel, not of the carcinomatous type, which would cause constriction and would be small. Because of its size the tumor had probably grown both within and outside the bowel, invading the mesentery. Myoma, fibroma and lipoma are possibilities but the more frequent variety is lymphoma, perhaps of the Hodgkin's type. We have no further evidence of that, however. Perhaps if an expert hematologist had seen a smear he would have been able to tell more accurately, but I should say that this was a lymphoblastoma or lymphosarcoma of the jejunum.

DR. TRACY B. MALLORY: Dr. Simeone, will you give the operative findings?

DR. F. SIMEONE: At operation this patient had a large mass about 9 or 10 cm. in diameter, which was stony hard to palpation and, on the surface, appeared white. The mass was freely movable in the horizontal plane from right to left and back again, but could not be moved in the vertical plane. It could not be delivered into the wound. The mass had infiltrated the mesentery to a great extent. On tracing it down it was found to extend to the origin of the midcolic artery and for that reason we believed that it was certainly incurable, but we wanted to make every effort to remove the tumor

FIGURE 1. *X-Ray Film Showing Displacement of the Proximal Transverse Colon by a Midabdominal Mass.*

because one of the symptoms was the big mass in the abdomen, which disturbed the patient. The resection carried us into the pancreas and to the midcolic artery. We thought it was unwise to go farther, in spite of the fact that tumor tissue appeared to extend beyond that point. Because of the amount of mesentery that had to be removed it was necessary to resect the entire jejunum and to mobilize the third portion of the duodenum from beneath the ligament of Treitz. An end-to-end anastomosis was done between the duodenum and the ileum.

FIGURE 2. *X-Ray Film of the Upper Jejunum. The arrow points to the ulceration in the jejunum.*

CLINICAL DIAGNOSIS

Carcinoma of jejunum.

DR. SOWLES'S DIAGNOSIS

Lymphosarcoma of jejunum.

ANATOMICAL DIAGNOSIS

Adenocarcinoma of jejunum.

PATHOLOGICAL DISCUSSION

DR. MALLORY: This clinic today is a good example of what poor judgment it would sometimes be to make the correct diagnosis. The last case* should have been a benign tumor but proved to be a malignant one. This case showed a typical ulcerative carcinoma, which had extended very deeply into the mesentery and had also become adherent to and invaded an adjacent loop of small bowel.

*Case records of the Massachusetts General Hospital (Case 32211). *New Eng. J. Med.* 234:701-703, 1946.

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THE MEDICAL CENTER FOR CHILDREN AT THE CHILDREN'S HOSPITAL

The *Journal* notes with interest the recent announcement of the proposed expansion of the Children's Hospital leading to its transformation into the Medical Center for Children.

During its seventy-seven years of service the Children's Hospital has earned world-wide recognition for the quality of its medical and surgical work, for its research and for its training of medical students, physicians, nurses, physiotherapists and other workers especially interested in the problems of early life. To its wards have been brought young patients from all over New England and indeed from distant places, both in this country and overseas. Its graduates are active in the practice of their specialties throughout the world.

The increased demands on the resources of the hospital, particularly from the New England area, the obsolescence of many of its buildings, and the opportunity for expansion of existing services and the creation of new units afforded by the constant effort of the staff to improve its work have led to the development of the Medical Center for Children. The heart of the plan, of course, is the Children's Hospital, with new and expanded facilities for the care of all aspects of disease in early life. To bring to older children, whose problems have long been neglected, all the medical, surgical and psychiatric assistance that will be available to younger patients, a new division of the Children's Hospital, a unit for adolescents, will be added. A neurologic institute, embracing the fields of neurology, neurosurgery and psychiatry, a greatly expanded infectious-disease division, with additional provisions for the care of patients with poliomyelitis, a cancer center and a greatly enlarged and completely equipped division of pediatric research form parts of the new plan. To assist in the most economical and efficient manner the doctors of New England who deal with young patients, a series of diagnostic clinics will be established. Similar facilities will be available in the several laboratory divisions that are designed to act as centers for consultation in chemical, clinicopathological, bacteriological and pathological aspects of pediatrics. In the field of preventive medicine, a health unit founded to bring to the problems of the healthy child from birth through adolescence all the specialized talent available in such a medical center should do much to raise the level of mental and physical health in New England and, in the course of time, to reduce the burden of disease.

To enable the Medical Center for Children to render the greatest possible service to the New England community, plans are now under way for appropriate affiliation with physicians and pediatric wards of general hospitals throughout this area, in extension of the unique relation that has existed for so long between the doctors in this region and the Children's Hospital. This affiliation, it is planned, will include not only arrangements for the

care of specialized medical and surgical cases but also provision for the training and postgraduate education of practicing physicians, interns, nurses and other workers with special interest in pediatrics.

The essence of the Medical Center for Children is the gathering under one roof, for a combined attack on the manifold problems of health and disease of the infant, the child and the adolescent, doctors and medical workers whose major interests concern these age groups, with the aid of all the technics and specialized knowledge available in the medical world today.

The long history of distinguished contributions of the Children's Hospital to the various medical, surgical and laboratory aspects of disease of infancy and childhood is a guarantee of the success of this ambitious but wholly logical and practical plan. The *Journal* extends its best wishes to the Board of Trustees and to the Medical Staff of the Children's Hospital in their great undertaking.

CHEMISTRY OF PENICILLIN

In sharp contrast to the many useful functions performed during the war by Government-supported research, there was at least one undesirable feature of that program, namely, the withholding of newly acquired information. The chief purpose of that secrecy—to keep from the enemy all information that may give him aid and comfort — is quite understandable. On the other hand, this policy has often served to channel information within a relatively small group of workers and away from others, perhaps of equal competence, who might have put that information to good use. The disadvantages and dangers of this sort of secrecy and the importance of the freedom to publish the results of scientific research are well recognized, and a vigorous attempt to preserve that freedom in any program of federally supported research is being made by a number of leading scientists.

Now that the war is over, there is still a good deal of scientific information that is being considered as "secret" or "classified" and is not yet published or otherwise freely available. Although there may be some justification for this attitude in matters of immediate national defense, such as those involved in the methods of manufacturing atomic bombs, many facts concerned with health are also included. Some of the latter information is gradually coming to light. Because of the vast amount of research and the colossal number of papers that are undoubtedly available for publication on certain subjects, the Committee on Medical Research and other similar bodies have wisely adopted the policy of summarizing the results of research in certain vital fields in advance of the publication of the detailed results.

One of the first of these summaries concerns the chemistry of penicillin.* Many of the most outstanding among the research laboratories in the United States and Great Britain participated in the program. Although the exact structural formula of penicillin is still undetermined, and its synthesis has not yet been accomplished, some of the most significant aspects of its chemistry have been determined beyond reasonable doubt.

Previously published reports had indicated that several forms of penicillin exist, each of which may be derived from cultures of the same strain of Penicillium. The usual form is penicillin G, which is the chief active ingredient of the commercial preparations now available. In addition, penicillin X has been described, and limited experimental and clinical studies of its use have been made in this country. Another form, penicillin F, has also been recognized in this country and in Great Britain. According to the joint report by the Office of Scientific Research and Development and by the Medical Research Committee of Great Britain, the forms of penicillin known as F, G and X in this country and as I, II and III, respectively, in Great Britain, as well as a new form, K-penicillin, all have the empirical formula $C_9H_{11}O_4SN_2R$. The differences depend on the structure of R. In penicillins G and X, for example, the structures of R are quite closely related, being benzyl in the former and parahydroxybenzyl in the latter. Each different form can, of course, be made into various metallic salts or esters. The varieties of penicillin in general

*Committee on Medical Research, O. S. R. D., Washington, and the Medical Research Council, London. Chemistry of penicillin. *Science* **102**: 627-629, 1945.

clinical use are the sodium and calcium salts of penicillin G.

These findings alone are of considerable general interest. Other features of the chemistry of penicillin, more important perhaps to the chemist and pharmacologist than to the clinician, are also published for the first time. According to the information thus far revealed it seems highly doubtful that synthetic penicillin will be forthcoming in the immediate future. Even should such synthesis be accomplished in the laboratory, it does not follow that the large-scale production of synthetic penicillin would be economically feasible. It is well known that the commercial manufacture of such simple substances as acetic and citric acids and various simple alcohols is best accomplished by fermentation methods, and the same may prove to be the case with penicillin.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

LANDESMAN — Henry M. Landesman, M.D., of Boston, died May 22. He was in his sixty-second year.

Dr. Landesman received his degree from the College of Physicians and Surgeons, Boston, in 1917. He was a fellow of the American Medical Association and a member of the Massachusetts Association of Examining Physicians and the South End Medical Club.

His widow and a daughter survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR APRIL, 1946

Résumé

Diseases	April 1946	April 1945	Seven-Year Median
Anterior poliomyelitis	2	2	1
Chancroid	2	2	*
Chicken pox	1751	963	1181
Diphtheria	16	25	13
Dog bite	1251	1286	945
Dysentery, bacillary	10	0	2
German measles	1190	195	398
Gonorrhea	453	463	355
Granuloma inguinale	0	0	*
Lymphogranuloma venereum	1	4	*
Malaria	35	124	3
Measles	8612	732	4012
Meningitis, meningococcal	14	16	16
Meningitis, Pfeiffer-bacillus	3	2	2
Meningitis, pneumococcal	5	7	6†
Meningitis, staphylococcal	0	0	0†
Meningitis, streptococcal	0	0	1†
Meningitis, other forms	2	2	2†
Meningitis, undetermined	4	5	5†
Mumps	924	2431	1482
Pneumonia, lobar	128	322	360
Salmonella infections	3	6	5
Scarlet fever	824	1422	1422
Syphilis	454	342	423
Tuberculosis, pulmonary	245	206	224
Tuberculosis, other forms	12	7	18
Typhoid fever	3	2	3
Undulant fever	9	2	3
Whooping cough	477	568	625

*Made reportable December, 1943.

†Four-year average.

Comment

Diseases reported at figures above the seven-year median included chicken pox, bacillary dysentery, German measles, pulmonary tuberculosis and undulant fever.

Measles was also reported above the seven-year median, reaching a peak since 1906 that is second only to the record year of 1934.

Reported at figures below the seven-year median were mumps, scarlet fever and whooping cough.

Record low figures were reported for lobar pneumonia.

The first case of animal rabies since February, 1944, was reported from the central part of the Commonwealth. The animal was a recent arrival from a nearby state.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Deerfield, 1; Springfield, 1; total, 2.

Diphtheria was reported from: Andover, 1; Arlington, 1; Boston, 5; Chelsea, 1; Cushing General Hospital, 1; Falmouth, 2; Haverhill, 1; Malden, 1; Millville, 1; Taunton, 1; West Newbury, 1; total, 16.

Dysentery, bacillary, was reported from: Belmont, 1; Boston, 2; Cambridge, 3; Lowell, 1; Peabody, 1; Wrentham (State School), 2; total, 10.

Encephalitis was reported from: Chatham, 1; total, 1.

Malaria was reported from: Amherst, 1; Boston, 8; Cambridge, 1; Chelsea, 1; Fall River, 2; Gardner, 1; Haverhill, 1; Hingham, 1; Lawrence, 1; Lynn, 1; Nantucket, 1; New Bedford, 1; Peabody, 1; Salem, 1; Southbridge, 1; Stoughton, 1; Waltham Regional Hospital, 2; Warren, 2; West Springfield, 1; Woburn, 1; Worcester, 5; total, 35.

Meningitis, meningococcal, was reported from: Boston, 4; Fall River, 1; Lee, 1; Ludlow, 1; Malden, 1; Medford, 1; Millbury, 1; Quincy, 1; Salem, 1; Wayland, 1; Worcester, 1; total, 14.

Meningitis, Pfeiffer-bacillus, was reported from: Cohasset, 1; Haverhill, 1; Saugus, 1; total, 3.

Meningitis, pneumococcal, was reported from: Abington, 1; Brockton, 1; Gloucester, 1; Natick, 1; Pittsfield, 1; total, 5.

Meningitis, other forms, was reported from: Boston, 2; total, 2.

Meningitis, undetermined, was reported from: Dalton, 1; Framingham, 1; North Adams, 1; Springfield, 1; total, 4.

Salmonella infections were reported from: Malden, 1; Northampton, 1; Springfield, 1; total, 3.

Septic sore throat was reported from: Arlington, 1; Belmont, 1; Boston, 4; Everett, 1; Greenfield, 1; Haverhill, 1; Newton, 2; Norfolk, 1; Westford, 2; Weston, 1; total, 15.

Tetanus was reported from: Pittsfield, 1; total, 1.

Trichinosis was reported from: Adams, 1; Boston, 4; total, 5.

Typhoid fever was reported from: Boston, 1; Chicopee, 1; Waltham, 1; total, 3.

Undulant fever was reported from: Belmont, 1; Boston, 1; Deerfield, 1; North Andover, 1; Oxford, 1; Quincy, 1; Southbridge, 1; Tewksbury, 1; Yarmouth, 1; total, 9.

REPORT OF MEETING

NEW ENGLAND ROENTGEN RAY SOCIETY

The New England Roentgen Ray Society held its last meeting of the season on Friday, May 17, 1946. The following officers were elected for the ensuing year: president: Dr. Samuel A. Robins; vice-president: Dr. Stanley A. Wilson; secretary: Dr. George Levene.

The second George W. Holmes Annual Lecture was delivered by Dr. Arthur C. Christie, of Washington, D. C. Dr. Christie's subject was "The First Fifty Years of Radiology: The elements which have contributed to its growth as a great medical specialty."

Dr. George W. Holmes and Dr. Christie were presented with the Society's silver colonial pitcher, which is emblematic of the occasion. Dr. Holmes received congratulatory telegrams from his former students in various parts of the country.

BOOK REVIEWS

Medical Gynecology. By James C. Janney, M.D. 8°, cloth, 389 pp., with 97 illustrations. Philadelphia: W. B. Saunders Company, 1945. $5.00.

There have been those who say, "There is no such thing as medical gynecology." It is therefore all the more gratifying that an author of Dr. Janney's distinction should devote his new volume to medical office gynecology. It has developed largely from his experience in clinical teaching for the past quarter century, and aims to aid the student in correlating his didactic lectures with his experience in the gynecologic clinic and to provide refresher material for the general practitioner. It follows the standard pattern of other texts — history and examination, symptoms, physical findings, laboratory tests and special examinations, and gynecologic treatments of a medical nature. There are chapters dealing with certain controversial subjects, such as abortion, contraception, illegitimacy and sterilization. Finally, there is an admirable brief section on the alternatives of radiation or surgery in the treatment of gynecologic malignant tumors and of intractable uterine hemorrhage of nonmalignant origin. The book is illustrated with well-chosen cuts and has a classified select bibliography of forty-one titles.

Trauma in Internal Diseases: With consideration of experimental pathology and medicolegal aspects. By Rudolf A. Stern, M.D. With a foreword by Francis C. Wood, M.D. 8°, cloth, 575 pp. New York: Grune and Stratton, 1945. $6.75.

It is a generally accepted principle of modern society that the workman injured through no fault of his own during the performance of his labors is entitled to compensation. The application of this principle is easy when the trauma is obvious, as, for example, when a fracture is sustained, but can be extremely difficult in the so-called "internal diseases." The wisdom of Solomon often could not decide the degree of aggravation of a duodenal ulcer resulting from employment in a certain occupation. In such cases it is the duty of the physician to advise and enlighten the court.

Those members of the profession who find themselves even occasionally on the witness stand will find this book invaluable. The author has thoroughly reviewed diseases of the heart, lungs, kidneys, gastrointestinal tract, endocrine glands and blood with regard to the role of trauma in etiology or aggravation. Some two thousand sources are listed in the bibliography. Most of these are references to cases that have been decided in court, and it is quite possible that this book will prove to be as useful to the lawyer as to the doctor.

American Red Cross First Aid Textbook. 12°, paper, 254 pp., with 264 illustrations. Philadelphia: The Blakiston Company, 1945. Sixty cents.

This book has been completely revised, with new illustrations and a rewritten text. It was prepared with great care under the direction of the Committee of Surgery, Division of Medical Sciences, National Research Council. In addition to revision of the chapters on surgery, the medical director of the American Red Cross and his staff fully revised the sections on first aid, water safety and accident prevention. Special chapters were contributed by authorities on injuries to the eye and on snake and other animal bites. The revision is by far the most complete since the book was first published in 1909, and with its splendid illustrations, clear text and adequate index, this volume should become a valuable adjunct to the work of the American Red Cross. In addition, it should appeal to the medical profession and should be in the hands of every first-year medical student. It will find a fitting place in many homes and factories. Every police officer and other public official who has to deal with acute emergencies should be thoroughly familiar with the contents of this book.

Mass Radiography of the Chest. By Herman E. Hilleboe, M.D., and Russell H. Morgan, M.D. 12°, cloth, 288 pp., with 93 illustrations. Chicago: The Year Book Publishers, Incorporated, $3.50.

One of the chief medical dividends of the late war was the vast experience in mass radiography of the chest. This monograph by two outstanding authorities on that subject is of timely interest and value, as the experiences and technics learned in the induction centers and service installations will undoubtedly be spread to large portions of the civilian population. This manual, as it may properly be called, was prepared for just such use, and it brings together the accumulated information on objectives, planning, equipment and technics, as well as valuable experiences in the procedures necessary to successful surveys of large numbers of people.

The authors lean over backward in the attempt to avoid the charge that such surveys may be interpreted as diagnostic examinations. Many of the common diseases and conditions involving the chest, heart and lungs can be identified quite accurately by the small-film technic. Quite correctly, however, they emphasize throughout the book the fact that their methods are primarily for screening purposes and that detailed clinical studies may be necessary in the positive and doubtful or borderline cases.

The chapter "Roentgen Diagnosis of the Chest" is particularly conservative and might well have been omitted, except that there is no similar primer for the uninitiated; it is to be hoped most sincerely, however, that the untrained will not attempt such an important public-health function.

This manual is of particular value to public-health officers, tuberculosis-control organizations, radiologists and all who are interested in the maximum efficiency and return from public-health expenditures.

NOTICES

ANNOUNCEMENTS

Dr. Warren D. Babb, having returned from military service, has resumed the practice of internal medicine at 40 Chestnut Street, Salem.

Dr. Samuel Bolan announces his return from service with the United States Navy and the reopening of his office at 555 High Street, West Medford.

Mr. William S. Brines took over the position of administrator of the Malden Hospital, Malden, on June 1.

Dr. Sidney C. Dalrymple, having been released from active duty with the Navy, announces the opening of an office for the practice of medicine at Middle Dyke Farm, Georgetown, Maine.

Drs. Channing Frothingham, George P. Denny and Victor G. Balboni announce the opening of offices for the practice of internal medicine at 101 Bay State Road, Boston.

Dr. Luke Gillespie, having been released from military service, has returned to the practice of obstetrics and gynecology at 1180 Beacon Street, Brookline.

Dr. Alfred M. Glickman announces his return from service with the United States Navy and the reopening of his office at 95 State Street, Springfield, for the practice of surgery.

Dr. Elwood O. Horne, having recently returned from military service, announces the opening of his office for the practice of general surgery at 36 Pleasant Street, Worcester.

Dr. Charles Schlosberg announces his return from military service and the resumption of pediatric practice at 483 Beacon Street, Boston.

Dr. Leslie H. Van Raalte announces the resumption of private practice limited to gynecology and obstetrics at 15 Lincoln Avenue, Wollaston.

(*Notices continued on page xix*)